HOSPITALS

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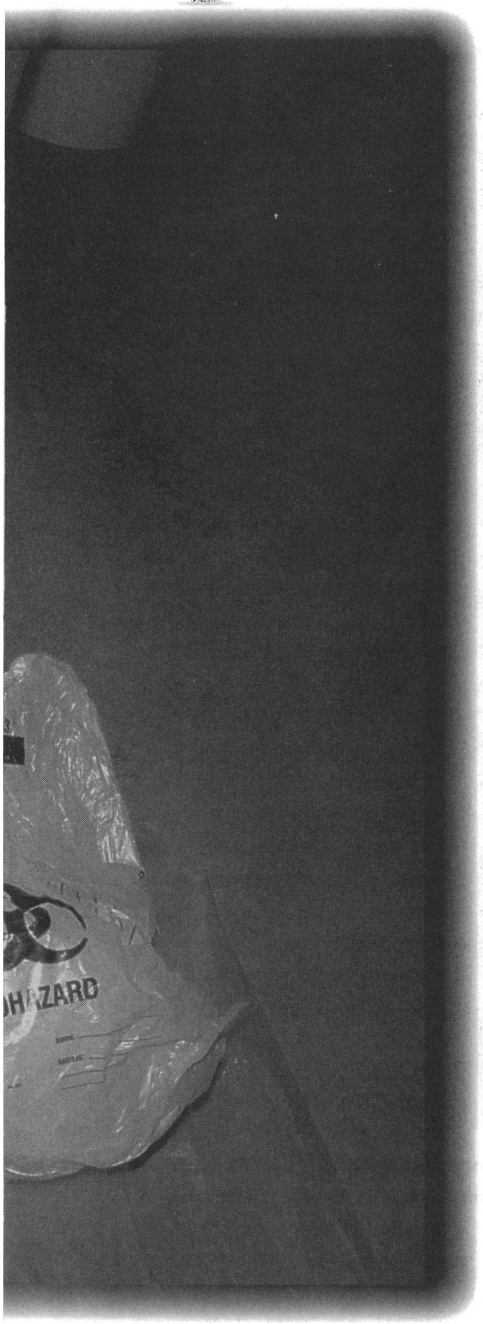
DIOXIN PREVENTION AND ME

& PLASTICS



SYNOPSIS

CHLORINATED DIOXINS and related compounds are extremely potent toxic substances, producing effects in humans and animals at extremely low doses. Because these compounds are persistent in the environment and accumulate in the food chain, they are now distributed globally, and every member of the human population is exposed to them, primarily through the food supply and mothers' milk. An emerging body of information suggests that dioxin contamination has reached a level that may pose a large-scale, long-term public health risk. Of particular concern are dioxin's effects on reproduction, development, immune system function, and carcinogenesis.

Medical waste incineration is a major source of dioxins. Polyvinyl chloride (PVC) plastic, as the dominant source of organically bound chlorine in the medical waste stream, is the primary cause of "iatrogenic" dioxin produced by the incineration of medical wastes. Health professionals have a responsibility to work to reduce dioxin exposure from medical sources. Health care institutions should implement policies to reduce the use of PVC plastics, thus achieving major reductions in medically related dioxin formation.

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In 1994, the U.S. Environmental Protection Agency (EPA) released its long-awaited "dioxin reassessment,"[1] which summarized an immense body of recent research on the toxicity, sources, and occurrence in the environment of the class of dioxin-like compounds. These substances, now globally distributed, are extremely potent toxic substances that produce a remarkable variety of adverse effects in experimental animals, fish, and wildlife, even at low levels of exposure. An emerging body of scientific evidence, presented in EPA's dioxin reassessment and elsewhere,[2] indicates that current "background" exposures to dioxin may represent a long-term, large-scale public health risk to people. Of particular concern are dioxin's impacts on reproduction, development, and immune system function.

In its reassessment, EPA also presented an inventory of dioxin sources,[3] which showed that medical waste incinerators were among the largest identified dioxin sources

Medical waste is collected in "red bags," one of which is opened here. Bags contain dioxin-releasing wastes, including gloves, tubing, bedpans, trays, and IV and infusion bags.

DICAL WASTE INCINERATORS

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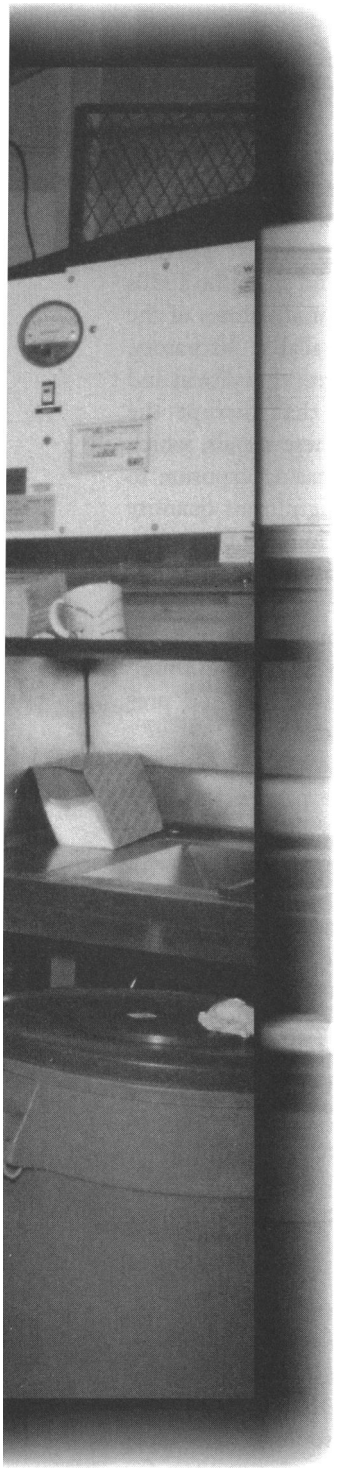
This "Groder" is typical of the medical waste incinerators installed in hospitals, medical schools, and laboratories. The EPA estimates that there are 2200 to 6700 medical waste incinerators in the United States. Regulations may soon require the "best available technology," reducing but not eliminating dioxin releases.

in the United States. Institutions dedicated to the prevention and cure of disease may thus turn out to be the greatest single contributor to a potentially major public health risk. Incinerators produce dioxin and dioxin-like compounds as products of incomplete combustion when chlorinated organic substances are burned. In medical waste, the predominant source of these compounds is polyvinyl chloride (PVC) plastic, an inexpensive and common polymer used for both medical and commercial products. Iatrogenic dioxin pollution can be largely eliminated by replacing PVC products with alternative materials.

Given the irony that health care facilities are a large source of these extraordinarily hazardous chemicals, health professionals have a responsibility to address these issues.



Health professionals should seek to implement PVC substitution policies in their institutions and in society at large. Ultimately a virtually chlorine-free hospital materials policy may become a realistic goal.

Global Distribution and Exposure

The group of dioxin-like compounds includes all substances similar in chemical structure and biological effects to 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). Among these are the polychlorinated dibenzo-p-dioxins (PCDDs) and the polychlorinated dibenzofurans (PCDFs) that have chlorine or bromine atoms substituted in the 2,3,7, and 8 positions; a number of coplanar polychlorinated biphenyls (PCBs); and many other halogenated, coplanar polynuclear aromatic hydrocarbons such as the polychlorinated biphenylenes, naphthalenes, and dibenzothiophenes.

Although we do not know the precise extent to which dioxin exposure has already affected human health, we can conclude with certainty that universal exposure to these compounds represents a risk to public health.

The terms "dioxin" and "dioxins" are used throughout this article as shorthand for "dioxin and dioxin-like compounds"; where research addresses specific members of the class, we refer to these specifically. These compounds are grouped together because they cause a similar spectrum of health effects via a common set of mechanisms, beginning with binding to a common intracellular receptor.[4] The toxicity of a mixture of dioxin-like compounds can be expressed in toxic equivalency (TEQ) units, a single term that represents the sum of the concentrations of each dioxin-like substance adjusted by its toxicity relative to that of 2,3,7,8-TCDD.

Two aspects of the environmental behavior of dioxins make them particularly troublesome. First, they are extraordinarily persistent: resisting physical, chemical, and biological degradation for decades and longer.[5,6] As a result, even dilute discharges accumulate in the environment over time, reaching particularly high levels in aquatic sediments and in the food chain. Because they are so long-lived and can be transported long distances through the atmosphere, dioxins are now distributed on a truly global basis.[7] Inuit natives of Arctic Canada, for instance, have among the highest body burdens of dioxins, furans, and PCBs recorded, due to a diet dependent on fish and marine mammals from a local food chain contaminated by atmospheric dioxin deposits generated by distant industrial sources.[8]

Second, dioxins are highly oil soluble but insoluble in water; thus they bioaccumulate in fatty tissues and are magnified in concentration as they move up the food chain. In species high on the food chain, dioxin body burdens are typically millions of times greater than the levels found in the ambient air, soil, and sediments.[9] Dioxins are also extraordinarily persistent in human tissues: estimated half-lives in humans are typically 5 to 10 years.[10]

At the apex of the food chain, the human population is particularly contaminated. A spectrum of dioxins has been identified in the fat, blood, and mother's milk of the general populations of the United States and Canada.[7] Virtually all human exposures to these compounds occur through the food supply, particularly consumption of animal products.[11] Significant quantities are passed from mother to child during the most sensitive stages of development: across the placenta and via mothers' milk.[7] The daily PCDD/PCDF dose received by the average nursing infant in the United States is 10 to 20 times greater than the average adult exposure.[4] A nursing infant thus receives about 10% of the entire lifetime exposure to these compounds during the first year of life.[1]

Health Effects of Dioxin

Biochemical studies have shown that dioxins act as powerful "environmental hormones." Much like natural hormones, these lipophilic substances cross cell membranes and bind to a receptor protein in the cytoplasm, the so-called Ah (aryl hydrocarbon) receptor. The dioxin:Ah receptor complex is then transported to the nucleus, where it binds specific DNA sequences, activating the transcription of genes whose products are involved in a wide range of biological functions. Unlike a natural hormone, however, dioxin resists metabolic degradation and has an extraordinarily high affinity for its receptor.[4] Tiny doses of these "false signals" can thus have powerful effects on processes regulated by endocrine mechanisms, including proliferation and differentiation of cells and the reproduction, development,

metabolism, and immune function of organisms.

In laboratory animals, exposure to dioxins, particularly 2,3,7,8-TCDD, has been associated with a remarkable variety of toxicological effects. (See Table 1.) Some of these effects have occurred at extraordinarily low doses. (See "Effects of Dioxin in Laboratory Animals and Humans" on page 306.)

In humans, our direct knowledge of dioxin toxicity comes from epidemiological studies of people exposed to relatively large amounts of dioxin in the workplace or due to industrial accidents for which exposures are unknown or difficult to reconstruct. Nevertheless, it is clear that dioxin causes cancer in humans, and the evidence is mounting that dioxin exposure has had adverse impacts on human reproduction and infant development as well. (See "Effects of Dioxin in Laboratory Animals and Humans" on page 306.)

Dioxin Poses a Global Health Risk

Every member of the general population, from the moment of conception until death, is now exposed to dioxin due to ubiquitous contamination of the food supply. Several lines of evidence suggest that these "background" exposures may pose significant public health risks. First, there does not appear to be a safe or "threshold" dose below which no health effects occur.[12] In fact, for those biochemical effects that have been investigated, the available data indicate a dose-response relationship that is linear or even supralinear.[13] Thus, low doses cannot be assumed safe.

The "background" exposures and body burdens of PCDD/PCDF in the general human population are approaching levels that clearly produce biological effects in laboratory animals.

Second, the "background" exposures and body burdens of PCDD/PCDF in the general human population are approaching levels that clearly produce biological effects in laboratory animals. Two interpretive reviews of the body of toxicological literature on low-dose dioxin exposures have concluded that current human body burdens are equal to or within one or two orders of magnitude of the levels that produce metabolic, reproductive, developmental, and immunological effects in laboratory animals.[6,14] This information suggests that at least part of the general human population now experiences health risk due to dioxin exposures.

Finally, it is clear that levels of dioxins in the environment have already caused large-scale effects on wildlife populations, particularly fish-eating birds and marine mammals. The most severe effects appear to be endocrine-mediated impairment of development, reproduction, and neurological and immune function.[15-20] If dioxin levels are high enough to cause these effects in wildlife, then humans—who are high on the food chain but have slower generation times—are likely to be at risk as well.

It is possible that universal exposure to dioxins has already had large-scale impacts on human health. For instance, incidence rates for many types of cancer have been increasing internationally for several decades.[21,22] Further, several authors have reported a global trend since the 1950s of falling sperm counts and increasing abnormalities of the human male reproductive tract.[23-25] Available laboratory, wildlife, and epidemiological studies suggest that dioxin and other persistent synthetic pollutants that disrupt the endocrine system may be one cause of these trends, which have occurred during the period of increasing exposure to compounds that were not produced in significant quantity before World War II.[25,26] These observations do not establish dioxin pollution as the cause of trends in sperm counts or in cancer rates, but they suggest a sound basis for concern and preventive action.

Although we do not know the precise extent to which dioxin exposure has already affected human health, we can conclude with certainty that universal exposure to these compounds represents a risk to public health. Any further increase in exposure must be expected to increase that risk and/or the severity of toxic effects.

Dioxin Sources: The Importance of Iatrogenic Pollution

According to EPA's 1994 inventory, medical waste incinerators (MWIs) were the largest known source of dioxins in the United States.[3] Combustion-related industrial processes accounted for over 90% of the dioxins and furans entering the environment from known sources. (See Table 2.) This sector included MWIs, followed by incinerators for municipal and hazardous waste, metal smelters, and combustion of automobile and truck fuels. EPA's inventory of U.S. dioxin sources was preliminary, and further investigations are likely to lead to quantitative revisions.

The dominant position of health care institutions among dioxin sources is striking. EPA estimated that the nation's 6700 medical waste incinerators emitted a total of 5100 grams (TEQ) of PCDD/PCDF per year. Because of uncertainty in emissions estimates, EPA provided a possible range for dioxin air emissions from MWIs of 1600 to 16,000 grams (TEQ) per year. MWIs also release additional

but unknown quantities of dioxin in ash, slag, and scrubber effluent. According to EPA's inventory, MWIs accounted for 45% of dioxin emissions from all identified sources and about one-fourth of the total PCDD/ PCDF flux that enters the U.S. environment (25,000 grams [TEQ] per year). The American Hospital Association has disputed these conclusions,[27] but other dioxin-release inventories have confirmed EPA's finding that MWIs are major dioxin sources.[28,29] One analysis[28] found that MWIs are the largest source of PCDD/PCDF deposits in the Great Lakes, while another[29] found that MWIs were second only to municipal waste incinerators as sources of PCDD/PCDF air emissions in the United States. Whether MWIs are the largest dioxin source or merely a major source, they clearly represent a health and environmental problem as well as a challenge for the professionals and institutions who manage and contribute to these sources.

Health hazards may be significant for local populations living near MWIs. According to the California Air Resources Board, PCDD/PCDF emissions from such facilities pose lifetime cancer risks ranging from 1 to 246 per million to persons in neighboring communities, considering only a few of many possible exposure pathways.[30] MWIs are also the largest known source of mercury emissions in the United States, and they are important sources of cadmium and lead emissions as well.[31,32] Additional hazards may be posed by releases of respirable particulate matter, hydrogen chloride, and other products of incomplete combustion, such as carbon tetrachloride, vinyl chloride, polychlorinated biphenyls, chlorobenzenes, chloroform, and chlorophenols.[30,33-35]

Most medical waste incinerators are small (capable of burning less than 150 pounds per hour) to medium-sized (capable of burning up to 1000 pounds per hour) units located on-site at hospitals and other medical facilities. An estimated 60% of the nation's medical waste is disposed of in such units.[36] In addition, approximately 150 commercial incinerators accept medical wastes from a variety of sources. These units, which have average capacities four to five times larger than on-site hospital incinerators,[3] burn an estimated 20% of the nation's medical waste. The remaining 20% is autoclaved[36] and ultimately ends up in landfills. The use of commercial incinerators is expected to increase as more stringent regulations lead to the shutdown of many uncontrolled on-site MWIs.

Plastics comprise as much as 30% of medical waste weight, in contrast to only 3% to 7% of municipal trash.[37] For the last decade, medical waste generation has increased rapidly as disposables have replaced reusables.[38] In the decade from the late 1970s to the late 1980s, the plastics content of medical waste increased from 10% to 30%.[39]

Table 1. Toxicological effects of dioxin and dioxin-like compounds

Modulation of hormones, receptors, and growth factors
Steroid hormones and receptors (androgens, estrogens, and glucocorticoids)
Thyroid hormones
Insulin
Melatonin
Vitamin A
EGF and receptor
TGF-a and TGF-b
TNF-a, IL1b, c-Ras, c-ErbA
Carcinogenesis
Immune system effects
Suppression of cell-mediated and humoral immunity
Increased susceptibility to infectious challenge
Autoimmune response
Developmental impacts
Birth defects
Fetal death
Impaired neurological development and subsequent cognitive deficits
Altered sexual development
Male reproductive toxicity
Reduced sperm count
Testicular atrophy
Abnormal testis structure
Reduced size of genital organs
Feminized hormonal responses
Feminized behavioral responses
Female reproductive toxicity
Decreased fertility
Inability to maintain pregnancy
Ovarian dysfunction
Endometriosis
Other effects
Organ toxicity (liver, spleen, thymus, skin)
Diabetes
Weight loss
Wasting syndrome
Altered glucose and fat metabolism

NOTE: This list includes effects identified in laboratory animals, humans, or cultured cells.
SOURCE: Adapted from references 1, 4.

Ethical Issues for Health Professionals

Health professionals have societal responsibilities arising from their status, knowledge, and skills, including an obligation to alert those at risk to the presence of a danger to their health.[40] Health professionals' commitment to act on behalf of the general public welfare is manifested in responsibilities such as reporting certain infectious diseases to proper authorities and notifying motor vehicle departments of medical conditions that might compromise an individual's ability to drive.

A concern for the health of the public has been established since the industrial revolution as an important responsibility of the medical profession,[40] although the proper extent of this commitment remains controversial. For public health workers, a commitment to disease prevention lies at the very foundation of their field.

Given that global dioxin pollution has profound implications for the health of individuals and communities and that medical waste contributes substantially to this pollution, health professionals bear a responsibility to act to prevent dioxin exposure. In 1982, Cassel and Jameton put forward a formal argument for the involvement of physicians in the prevention of nuclear war.[41] We believe that a similar argument should compel health professionals to act to prevent exposures to dioxin and other toxic substances.

Health professionals may be able to treat some of the health conditions caused by dioxin but cannot cure them.

Table 2. Major sources of dioxin and dioxin-like compounds in the United States, 1994

Dioxin source	Dioxin releases (grams [TEQ]/year)[a] Median estimate	Range
Medical waste incinerators	5100	1600 – 16,000
Municipal waste incinerators	4800	2110 – 10,700
Hazardous waste incinerators/ cement kilns[b]	409	120 – 1,200
Pulp mills	363	256 – 504
Wood burning	360	113 – 1,063
Secondary copper smelters	230	74 – 740
Vehicle fuel combustion	86	27 – 274
Forest fires	86	27 – 270
Sewage sludge incineration	23	10 – 52
Secondary lead smelting/ refining	1.6	0.7 – 3.5
Chemical manufacturing	NA	NA
Home and building fires	NA	NA
Ferrous metal smelting/ refining	NA	NA

SOURCE: Reference 3.

[a]Sum of releases to all media from each source. Ranked by median estimate.

[b]Based on EPA's estimate that dioxin emissions from cement kilns burning hazardous waste are one order of magnitude greater than from kilns burning traditional fuels. See reference 3.

NA=Not available.

When cure is not possible and prevention is effective, prevention is ethically required. It is thus incumbent upon those in the fields of medicine and public health to work to avoid dioxin exposures by preventing further environmental contamination by dioxin—particularly that caused by the health industry itself. Since dioxin pollution can be reduced considerably through changes in medical waste and incineration practices, health workers should strive to prevent further dioxin exposure by influencing the materials management practices of hospitals and other health-care institutions.

Two Frameworks for Policy

What approach will yield the most substantial reduction in environmental releases of dioxin? In general, two strategies are available: one emphasizes pollution control, the other pollution prevention.

The pollution control strategy, the traditional approach underlying most current policy in the United States, focuses on reducing emissions through improved management of the sources of dioxin. Technologically, this approach relies on enhanced pollution control technologies that capture pollutants before they are released into the environment. Such "end-of-pipe" methods include scrubbers, precipitators, filters, and effluent treatment systems. Captured pollutants must then be treated or disposed of by other means. Ash and dust from incinerator pollution control devices are typically placed in landfills.

In contrast, prevention strategies seek to avoid the generation of dioxin altogether, thus eliminating discharges into the environment. The ultimate preventable source of dioxin generation is the materials that supply chlorine for incorporation into dioxin, for example, during incineration. Although a number of potential pathways for dioxin formation have been proposed, all require the following: a source of chlorine, a source of organic matter, and a thermally or chemically reactive environment in which these materials can combine.[3] The presence of chlorine donor material turn industrial processes into dioxin sources. Thus these materials are the appropriate focus for preventive efforts. Prevention strategies thus rely on "front-end" alterations such as changing production processes, substituting input materials, and reformulating products.

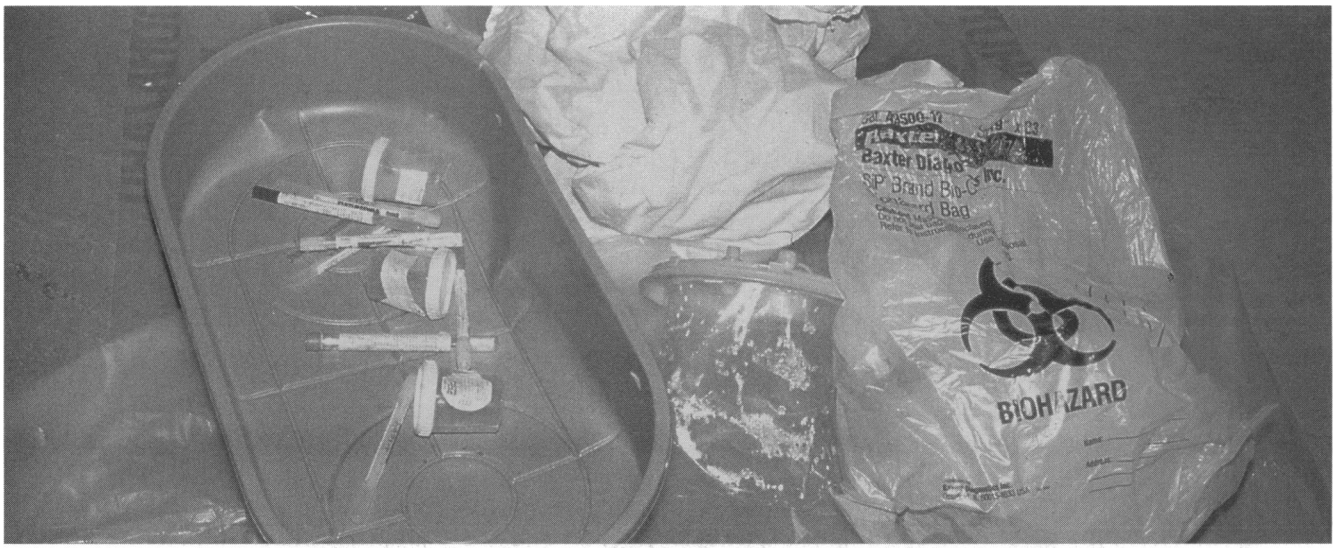
Ethical issues should also affect the choice of regulatory strategy. Control-oriented regulations, because they do not eliminate the production of hazardous pollutants, must establish an "acceptable" level of pollutant discharge and exposure, typically using quantitative risk assessments to predict possible health impacts. The health impacts of dioxin, however, are not completely characterized, particularly at low doses. New releases to the environment at levels currently considered acceptable continue to permit involuntary human exposures on a large scale until the toxicity is fully understood. This regulatory paradigm creates, in effect, a vast program of human experimentation without informed consent.

In contrast, a prevention approach errs on the side of caution by anticipating environmental damage and changing production processes to avoid generation of hazardous pollutants. Unlike reactive environmental policies, which intervene only after damage has occurred and risks are fully quantified, this approach is based on a precautionary principle: in the face of continuing uncertainty about the exact nature and magnitude of associated injury, we must seek to anticipate and prevent environmental damage before it occurs. The principle applies fundamental tenets of public health practice—the primacy of prevention, and "first do no harm"—to environmental policy. The American Public Health Association has built on this analysis to argue for the precautionary phase-out of the class of chlorinated organic chemicals.[42-45]

Table 3. Costs of medical waste disposal methods

Method	Cost ($/lb)
On-site autoclave & shred	0.05 – 0.10
Microwave & shred	0.07 – 0.10
Off-site autoclave & shred	0.17
On-site incineration (controlled)	0.30
Off-site incineration (controlled)	0.50

SOURCE: References 30, 31.



Limitations of Control Strategy for Iatrogenic Dioxin Pollution

A pollution control strategy for medical waste incinerators would focus on improving incinerator technology to reduce dioxin emissions. In 1995, EPA proposed new regulations under the Clean Air Act that embody such an approach.[32] These rules would require MWIs to install pollution control equipment—specifically fabric filters and carbon injection units—and to improve combustion conditions to meet minimum requirements for furnace temperature and residence time (the period during which the burning material is kept inside the incinerator chamber). EPA predicts that full implementation of these rules will reduce MWI air emissions of dioxin by 99%, lead by 99%, cadmium by 97%, and mercury by 94%.

Given that global dioxin pollution has profound implications for the health of individuals and communities and that medical waste contributes substantially to this pollution, health professionals bear a responsibility to act to prevent dioxin exposure.

These rules promise to reduce but not eliminate the dioxin problem associated with MWIs. Under EPA's scenario, existing medical waste incinerators in the United States will still release dioxins and furans into the air.[32] MWIs will continue to contribute to the nation's dioxin burden and will pose health risks to local communities. The California Air Resources Board has estimated that MWIs with state-of-the-art pollution control continue to pose lifetime cancer risks in the one to three per million range to people in neighboring communities, estimated for just a few exposure pathways.[30]

Second, lower air emissions do not necessarily reflect reductions in the total environmental burden of dioxins because the control devices that EPA will require merely transfer pollutants from stack gas into ash and effluents, which are then disposed of in landfills or discharged into waterways.[31] An MWI in compliance with EPA's new regulations would deposit almost 20 times more dioxin in its ash than it would emit through its smokestack.[32] EPA has asserted that improved combustion conditions required under the proposed rules will reduce the total generation of dioxin, but there are reasons to be skeptical about this claim. An analysis of trial burns at a number of MWIs in California found that significant quantities of PCDD/PCDF are formed even when combustion conditions are good—that is, even with high temperatures, adequate residence time, and low emissions of carbon monoxide and total volatile hydrocarbons.[46,47]

Finally, pollution control devices are costly. EPA has estimated that its new regulations will more than double the cost of medical waste disposal. Costs at existing incinerators are expected to increase from $168 per ton to $390 per ton. EPA estimates that its new rules will raise the total nationwide expenditure on medical waste incineration from the current annual level of $340 million to $754 million.[32]

Thus, while the pollution control approach can, at some cost, reduce dioxin releases at individual locations, this framework cannot effectively reduce the total environmental burden. Dioxin contamination is qualitatively different from the conventional image of pollution, which is localized in space and time and can be addressed with the add-on technology of pollution control. Dioxin pollution is characterized by persistent global contamination leading to universal,

Effects of Dioxin in Laboratory Animals and Humans

Several studies indicate that dioxin can exert its toxic effects at extremely low doses. In primates, for instance, long-term exposure to 2,3,7,8-TCDD concentrations as low as five parts per trillion has been associated with impaired neurological development and endometriosis.[90,91] A single 64 nanogram per kilogram (ng/kg) dose of 2,3,7,8-TCDD delivered to pregnant rats on day 15 of gestation results in impaired sexual development of male offspring evident only at puberty, with impacts including altered reproductive anatomy, reduced sperm count, feminized hormonal responses, and feminized sexual behavior.[92]

Twenty-four-week TCDD doses as low as 0.3 ng/kg of body weight (BW) per week have caused immunological abnormalities in primates.[93,94] In rodents, one-time doses in the 1 to 100 ng/kg BW/day range have resulted in immunological abnormalities[95] and metabolic changes.[96,97] The recent finding that the genome of the HIV-1 virus contains regulatory sequences that bind the dioxin:AhR complex and activate transcription of viral genes is cause for concern that dioxin may also play a role in the expression of infectious disease.[98]

Dioxin is also an extremely potent carcinogen. Positive findings were reported in each of 18 studies that investigated the carcinogenicity of 2,3,7,8-TCDD, demonstrating that dioxin is a positive multisite carcinogen in both sexes in the rat, mouse, and hamster by all routes of exposure investigated. Tumors have been produced in nine organ systems at doses as low as 1 ng/kg BW/day, and no threshold level has so far been convincingly found for TCDD's carcinogenic activity.[99] EPA's current "acceptable daily intake" for PCDD/PCDFs, based on a one-per-million cancer risk, is 0.006 picograms (TEQ) per kilograms per day,[100] some 500 times lower than the "background" exposures to which the general U.S. population is now subject.

The effects seen in dioxin-exposed people are consistent with those seen in laboratory animals. Six recent well-designed epidemiological studies involving occupational and environmental exposures to PCDD/PCDFs have shown increased risks for cancers at numerous specific sites and for all cancers combined.[101–106] According to one review, "Epidemiological data from occupationally exposed workers now show accumulating and convincing evidence that exposures to TCDD are associated with several cancers in humans: respiratory, lung, thyroid gland, connective and soft tissue sarcoma, hematopoietic system, liver, and all cancers."[99] Dioxin is classified as a probable human carcinogen by the International Agency for Research on Cancer, the Environmental Protection Agency, and the National Institute for Occupational Safety and Health.

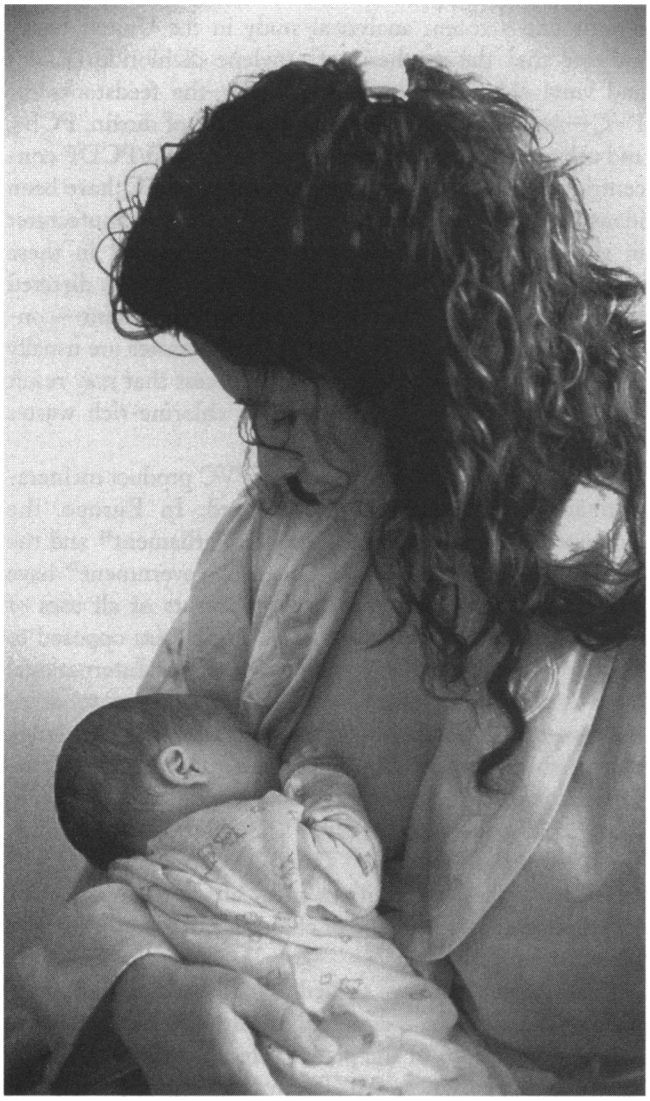
While the non-cancer effects of dioxin have received less attention, there is evidence that PCDD/PCDF exposure reduces male sex hormone levels and libido[1,107–109] and increases the risk of diabetes.[110,111] In human infants, several studies indicate dioxin-mediated effects on physical, cognitive, and sexual development.[112,113] Infants born to mothers who had consumed two to three meals per month of Great Lakes fish were underresponsive and hyporeflexic at birth and subsequently exhibited dose-dependent deficits in visual recognition memory and activity levels; these deficits were still present at follow-up at age 4. PCBs were measured in cord serum as a marker of total pollutant exposure, and the severity of developmental deficits correlated with PCB levels.[114–116] Similar PCB-related cognitive deficits were found in perinatally exposed infants in North Carolina, although the impairments present at birth and 12 months were not present at follow-up at 3 to 5 years.[117,118] Finally, there is evidence of alterations in thyroid hormone levels, increased incidence of intracranial hemorrhage, and immune suppression associated with lactational exposure to PCDDs, PCDFs, and PCBs in Europe and Arctic Canada.[119–121]

multigenerational exposure. Such a problem is of a scope similar to global warming and ozone depletion and warrants policy responses of similar scale.

Prevention of Iatrogenic Dioxin Pollution: The Role of PVC Plastic

Instead of focusing on improved management of dioxin-producing technologies, a prevention strategy for MWIs would seek to prevent dioxin formation altogether by changing the hospital waste stream. Polyvinyl chloride (PVC) plastic is the principal chlorine donor in medical waste.[35,48] It is used in packaging, infusion bags, tubing, bedpans, trays, gloves, and numerous other medical applications. According to one analysis for the city of New York, PVC gloves and IV-bags alone account for virtually all of the organic chlorine and over 80% of the total chlorine content of medical waste.[49]

PVC, which is 59% chlorine by weight, is the only major plastic that contains chlorine. Only a small amount of all PVC is used for medical applications, but the total production of PVC is immense: over 5.5 million tons per year in the United States, consuming about 30% of all chlorine produced by the U.S. chemical industry.[50] PVC is the most commonly used polymer in the medical device arena: an estimated 700 million pounds per year of PVC are used in



Significant quantities of dioxin and dioxin-like compounds are passed from mother to child during the most sensitive stages of development: across the placenta and via mother's milk. The daily PCDD/PCDF dose received by an average nursing infant in the United States is 10 to 20 times greater than the average adult exposure. A nursing infant receives about 10% of his or her entire lifetime exposure to these compounds during the first year of life.

medical devices in the United States, with an annual growth rate of 6.4%.[51]

PVC contributes about 80% of the organically bound chlorine found in municipal incinerators and about half of the total chlorine (organic plus inorganic).[52-54] While municipal waste contains only 0.5% PVC by weight, hospital red-bag waste—infectious wastes destined for incineration—contains an estimated 9.4% to 15% PVC, [55,56] while clear-bag wastes are as much as 18% PVC.[56]

As discussed below, laboratory, pilot, and full-scale studies confirm that the feed of chlorine—PVC in particular—leads to dioxin emissions. Of course, chlorine input is not the only factor that affects dioxin generation. Facility design, operating conditions, and the presence of catalysts also play important roles. These factors modulate the quantity of dioxin formed from chlorinated organic feedstocks. Several authors have noted that PVC, as the dominant source of organically bound chlorine in medical waste incinerators, is the primary cause of dioxin releases from these facilities.[29,31,37,46,55]

It has been suggested that the presence of chloride salts from biological and other materials will lead to dioxin formation in combustion devices, irrespective of the presence of chlorinated organic substances.[57] Thus the chemical industry has argued that there is no basis for restricting organochlorine-containing products.[58] Several lines of evidence indicate, however, that chloride salts are relatively unimportant dioxin precursors. First, no sound empirical data support the claim that combustion of chloride salts is a significant source of dioxin. The "chlorine-free" materials that produce dioxins when burned have never been tested for residues of organochlorine contaminants, which are now ubiquitous in air, water, and biological samples.[3] In contrast, several laboratory studies have found that burning PVC alone produces dioxin in concentrations up to the parts per million range.[59-61] Numerous laboratory studies have found that combustion of organochlorine-containing materials results in dioxin formation orders of magnitude greater than those associated with burning analogous substances containing chlorides but no organochlorines.[61-65]

The data for incinerators are similar. An ongoing investigation at a research incinerator at the University of Florida has found a clear relationship between the feed of PVC and emissions of dioxins.[35,66] The Danish Environmental Protection Agency has found that doubling the PVC feed to a municipal waste incinerator increases PCDD/PCDF emissions by 32%, while doubling the inorganic chloride content increases emissions by a much smaller margin.[52] A 1993 study for the Dutch Environment Ministry reported that reducing the PVC feed to a municipal waste incinerator led to a corresponding reduction in dioxin emissions, particularly when chloride concentrations were kept low.[53] When wood products containing PVC are burned in stoves and furnaces, dioxin PCDD/PCDF concentrations are up to three orders of magnitude greater than when unprocessed wood—which contains chloride—is burned.[65] Moreover, emissions data from a wide range of combustion facilities indicate a "clear dependence" of dioxin and furan emissions on the chlorine content of the wastefeed.[29]

Finally, the historical record underscores the importance of organochlorine combustion as a dioxin source and suggests that combustion of natural chloride-containing substances is not a significant source. PCDD/PCDF levels in the tissues of ancient humans exposed to a significant burden of wood smoke are no more than 1% to 2% of the amount found in modern humans. This observation is inconsistent with the theory that much of today's body burden could be due to natural sources such as forest fires.[3,7,66] Further, studies

of sediments in the Great Lakes[68] and of vegetation and soils in the United Kingdom[69] show that dioxin PCDD/PCDF levels remained very low throughout the 19th century. They began to climb toward their current concentrations only in this century, with the greatest increases after World War II. This pattern parallels the development and expansion of the chlorine chemical industry.

Several studies have found no relationship between incinerator dioxin emissions and PVC content of the waste feed. Two trial burns at municipal waste incinerators found no statistically significant association between PVC feed and dioxin emissions,[70,71] while a recent compilation of trial burn data from a large number of incinerators also found no relationship.[72] None of these investigations, however, controlled or adjusted for variations in other factors that are also known to affect dioxin emissions, such as operating conditions and wastefeed composition. A potential relationship between PVC and dioxin may thus have been masked by fluctuations in other factors. Indeed, an EPA reanalysis of the data from one of these studies[70] indicates that when combustion conditions were held constant, emissions of dioxins and furans tended to increase as PVC content of the waste rose.[73]

From this evidence, it appears that PVC is an important factor in dioxin formation in incinerators and that removal of PVC from the waste stream will significantly reduce the generation of dioxin.

Sunsetting Chlorinated Plastics in Hospital Supplies

Based on these considerations, PVC reduction emerges as a clear priority for dioxin prevention efforts. Strategies should focus on eliminating PVC from incinerator waste streams by substituting alternative materials. The Congressional Office of Technology Assessment and the Air and Waste Management Association agree that emissions of dioxin and hydrogen chloride from MWIs may be reduced by substituting non-halogenated plastics for halogenated plastics in hospital operations.[31,36]

Eliminating the use of PVC will provide other benefits not associated with air pollution control. First, it will reduce dioxin releases not just to air but also to waterways and land disposal facilities. Second, the stabilizers used in PVC products are another important source of MWI cadmium and lead emissions.[31,36,37] Substituting other materials for PVC will thus reduce these highly problematic releases. Third, because metal chlorides are more volatile than elemental metals or metal oxides, high chlorine content increases the release rate of toxic metals from an incinerator into the air; eliminating PVC feed to MWIs will thus reduce metals emissions.[74]

Finally, dioxins are produced when PVC is manufactured, so use of chlorine-free materials in place of PVC for medical products will prevent the dioxin pollution associated with the production of PVC. Numerous European reports and a recent analytical study in the United States indicate that the synthesis of ethylene dichloride (EDC) and vinyl chloride monomer (VCM)—the feedstocks for PVC—result in the formation and release of dioxin, PCBs, and other chlorinated by-products.[75-79] PCDD/PCDF concentrations as high as six parts per million TEQ have been identified in the wastes from one EDC/VCM manufacturer in the United States.[80] Contaminants produced in these processes are emitted to air, released in effluents, directed into PVC products, and—to a much greater extent—concentrated in tars and other wastes. These wastes are usually disposed of onsite by incineration, a process that may result in additional dioxin formation when chlorine-rich wastes are subject to incomplete combustion.

The recommendations to reduce PVC product incineration are by no means unprecedented. In Europe, the Enquete Commission of the German Parliament[81] and the Ecocycle Commission of the Swedish Government[54] have recently recommended complete phase-outs of all uses of PVC with short lifetimes and of all flexible (as opposed to rigid) PVC products. In North America, the International Joint Commission on the Great Lakes (IJC)—a treaty organization of the United States and Canada—has concluded that persistent toxic pollution is a hazard to health and the environment in the Great Lakes and has called for action to bring industrial discharges of 2,3,7,8-TCDD, 2,3,7,8-TCDF, and PCBs to zero.[82] The IJC has called on the governments of the United States and Canada to implement a timed phase-out of "the use of chlorine and organochlorines as industrial feedstocks."[83]

In summary, we have argued that global dioxin contamination is likely to be a significant health risk to human and animal populations of the planet. Medical waste is a major source of this pollution. An effective strategy to prevent the continued generation of dioxin is to remove organochlorine plastics from the medical waste stream. Reduction and eventual elimination of PVC use by the health care industry is an important first step in this process. Because medical institutions are major sources of dioxin, health professionals have a special responsibility to work to prevent dioxin pollution.

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A PROGRAM OF ACTION FOR HEALTH CARE INSTITUTIONS

To address dioxin pollution due to the presence of PVC in hospital waste, health professionals, institutions, and society at large must take steps to reduce and eventually eliminate the use of PVC in health care settings. This program requires institutions—and the individuals who manage or participate in them—to analyze the medical waste stream and identify PVC-containing products, to identify and implement alternative materials, and to improve medical waste disposal practices in general—without compromising hygiene or medical effectiveness.

Identify substitutes for PVC. Because PVC has useful properties and is a component of a great number of medical products, substituting chlorine-free alternatives presents a technical challenge. However, Klausbruckner reports that several European hospitals—including Vienna Ost hospital[84]—are reported to have virtually eliminated their use of PVC-containing products. The most appropriate substitute is specific to each product and is chosen from among chlorine-free plastics, glass, metal, or fiber products.[85] Some uses of PVC have been replaced with relative ease, including examination gloves, overshoes, aprons, mattress covers, diapers, wound dressings, and syringes.[51,85] Some uses of PVC are not particularly "medical," including office supplies, product packaging, building components such as pipes and flooring, fixtures, and furniture. Their replacement requires no consideration of medical efficacy or hygiene. For most applications that utilize rigid PVC, products made with metal, glass, or plastics such as polypropylene or polycarbonate or engineering thermoplastics can be substituted.[51]

Replacement of flexible PVC products presents more of a challenge. Several authors report that medically safe and effective substitutes for PVC made of chlorine-free polymers—such as polyolefins, ethyl vinyl acetate, polyester ethers, and block copolymers—are available for most uses for catheters, drainage bottles, collection bags, respiratory masks, and scalpels as well as infusion equipment, bottles, bags, connecting parts, and tubing.[51,85]

For a few PVC applications, including blood bags and infusion tubes for specific uses, no clearly demonstrated alternatives are yet in use. However, Wagener[51] argues that block copolymers consisting of alternating layers of hard and soft plastics offer superior performance and may be candidates for replacing PVC in these applications. These materials are biocompatible, meet technical specifications, and are available now from major manufacturers for IV infusion bags and other medical uses. As Wagener summarized, "The substitution of PVC destined for incineration because of infectious contamination can be readily accomplished with existing resins."[51]

Phasing out PVC thus appears to present real but not insurmountable technical challenges. Action to replace PVC-containing products should begin with those applications for which alternatives are most readily available while research, development, and testing continue on substitutes for other PVC products that are more difficult to replace. Although PVC is very inexpensive, the cost of substitution should not be prohibitive: for most medical products, the raw material cost accounts for only a small fraction of the total cost so even a doubling of the raw plastic costs would result in a relatively small increase in total price.[51]

Establish materials policy. A program to phase out PVC should be implemented in parallel with an overall materials policy designed to reduce both the quantity and toxicity of waste generated. Such a strategy, which may also reduce costs, begins with a waste audit at each hospital or facility to identify materials associated with toxic pollution during their manufacture, use, or disposal and to investigate the availability of "cleaner" substitutes. As a first step, hospitals should turn to safer processes than incineration for waste disinfection. Two recent papers report that autoclaving and microwaving are fully adequate disinfection technologies and that subsequent shredding can reduce volume by 60% to 80%.[30,31] The only wastes that are not suitable for treatment by these methods are pathological wastes (body parts and other tissue-derived wastes), which may be burned without significant pollution hazards. Autoclaving or microwaving, followed by shredding, costs 60% to 80% less than controlled incineration.[31] (See Table 3.)

At the same time, hospitals should seek to reduce the volume and toxicity of the waste they generate. Reducing volume focuses on the substitution of reusable materials for many disposable products, such as linens, gowns, draping, bedpans, food service equipment, and so on without compromising sanitary concerns.[38,86-88] One study suggested that substitution of reusable products for disposables and recycling of paper would result in a 93% reduction in surgical waste.[38] Reliance on reusables is reported to lower costs significantly, since costs to wash or disinfect items for reuse is less than that of purchasing new dispoable items.[31,38,86,89] For instance, one analysis found that a single teaching hospital saved over $100,000 a year by returning to reusable scrub suits and gowns in the operating room.[89]

What health professionals can do. The hospital materials and waste management policies suggested here must be brought to the attention of senior hospital management and defended as affordable and in the institution's interests. Health professionals are in a position to educate their peers, administrators, and hospital board members about dioxin prevention. These issues should be brought before professional associations of medical and public health workers. In turn, these organizations can participate in efforts to persuade governments to establish policies to reduce the use of PVC and the incineration of chlorinated materials.

Robert Visser, Greenpeace

Dioxins are highly oil soluble, accumulate in fatty tissues, and are magnified in concentration as they move up the food chain. Species and populations whose diets are rich in fish and marine mammals have among the highest body burdens of dioxins, furans, and PCBs.

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